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=> d his

     (FILE 'HOME' ENTERED AT 17:37:25 ON 20 DEC 2007)

     FILE 'MEDLINE' ENTERED AT 17:37:36 ON 20 DEC 2007

     FILE 'MEDLINE' ENTERED AT 18:02:24 ON 20 DEC 2007
L1        100858 S STROKE
L2        108644 S MEMORY
L3       2559837 S 1 AND 2
L4       2397179 S 1 (P) 2
L5           892 S L1 (P) L2
L6         58619 S DEMENTIA
L7         20677 S BIOMARKER
L8           256 S L6 AND L7
L9           231 S L6 (P) L7

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     FILE 'MEDLINE' ENTERED AT 00:42:52 ON 21 DEC 2007
L1         58619 S DEMENTIA
L2        160020 S DIFFICULT
L3           989 S L1 (P) L2
L4       1276369 S RAT
L5            10 S L3 AND L4
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=> d his

     (FILE 'HOME' ENTERED AT 15:31:47 ON 20 DEC 2007)

     FILE 'MEDLINE' ENTERED AT 15:32:15 ON 20 DEC 2007
L1         58604 S DEMENTIA
L2           148 S ALZHEIMERS
L3         58242 S ALZHEIMER
L4         58242 S L2 OR L3
L5       1320716 S GENERAL REVIEW/DT
L6         12040 S L5 AND L4
L7         10203 S STATE OF THE ART
L8            40 S L6 AND L7
L9            68 S L4 AND L7
L10            0 S L8 NOT L9
L11           28 S L9 NOT L8
L12        12040 S L5 AND L4
L13         9781 S UNPREDICT?
L14           12 S L13 AND L12
L15      2169219 S TREATMENT
L16         3143 S L15 AND L12

=> log hold
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      14.92      15.13

 SESSION WILL BE HELD FOR 120 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 15:48:58 ON 20 DEC 2007
1
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L16 ANSWER 89 OF 3143 MEDLINE on STN
AN 2007442275 MEDLINE
DN PubMed ID: 17646621
TI Rationale for transdermal drug administration in **Alzheimer** disease.
AU Oertel Wolfgang; Ross Joel S; Eggert Karla; Adler Georg
CS Philipps-University Marburg, Marburg, Germany.. oertelw@med.uni-marburg.de
SO Neurology, (2007 Jul 24) Vol. 69, No. 4 Suppl 1, pp. S4-9. Ref: 33
Journal code: 0401060. E-ISSN: 1526-632X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200708
ED Entered STN: 31 Jul 2007
Last Updated on STN: 17 Aug 2007
Entered Medline: 16 Aug 2007
AB Transdermal patches are used for the **treatment** of various diseases including neurologic and psychiatric disorders such as Parkinson disease (PD), major depression, and attention deficit hyperactivity disorder. They are believed to offer many advantages over conventional oral therapies. By providing smoother, continuous drug delivery and steadier plasma levels, patches may reduce the incidence of side effects, thus making optimal therapeutic doses easier to attain and potentially improving **treatment** efficacy and compliance. Drug delivery systems such as patches that are more patient- and caregiver-friendly may enable patients to continue **treatment** for longer periods and to attain greater, more sustained **treatment** benefits. To date, approved therapies for **Alzheimer** disease (AD), including cholinesterase inhibitors and memantine, are orally administered. Potential advantages associated with patches provide a therapeutic rationale to offer additional benefits in AD patients. Rivastigmine is well suited to patch administration because it is a small, potent molecule that is both lipophilic and hydrophilic. A rivastigmine patch has been developed and may provide a promising new approach to dementia therapy.

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L16 ANSWER 6 OF 3143 MEDLINE on STN
AN 2007676993 IN-PROCESS
DN PubMed ID: 17997702
TI Immunotherapy as **treatment** for **Alzheimer**'s disease.
AU Hawkes Cheryl A; McLaurin Joanne
CS Center for Research in Neurodegenerative Diseases, University of Toronto, Toronto, Ontario, Canada.. cheryla.hawkes@utoronto.ca
SO Expert review of neurotherapeutics, (2007 Nov) Vol. 7, No. 11, pp. 1535-48. Ref: 152
Journal code: 101129944. E-ISSN: 1744-8360.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20 Nov 2007
Last Updated on STN: 10 Dec 2007
AB **Alzheimer**'s disease (AD) is a progressive neurodegenerative disorder that is characterized pathologically by the deposition of beta-amyloid (A beta)-containing extracellular neuritic plaques, intracellular neurofibrillary tangles and neuronal loss. Much evidence supports the hypothesis that A beta peptide aggregation contributes to AD pathogenesis, however, currently approved therapeutic **treatments** do nothing to stop or reverse A beta deposition. The success of active and passive anti-A beta immunotherapies in both preventing and clearing parenchymal amyloid in transgenic mouse models led to the initiation of an active anti-A beta vaccination (AN1792) trial in human patients with mild-to-moderate AD, but was prematurely halted when 6% of inoculated patients developed aseptic meningoencephalitis. Autopsy results from the brains of four individuals treated with AN1792 revealed decreased plaque burden in select brain areas, as well as T-cell lymphocytes in three of the patients. Furthermore, antibody responders showed some improvement in memory task measures. These findings indicated that anti-A beta therapy might still be a viable option for the **treatment** of AD, if potentially harmful proinflammatory processes can be avoided. Over the past 6 years, this target has led to the development of novel experimental immunization strategies, including selective A beta epitope targeting, antibody and adjuvant modifications, as well as alternative routes and mechanisms of vaccine delivery, to generate anti-A beta antibodies that selectively target and remove specific A beta species without evoking autoimmunity. Results from the passive vaccination AD clinical trials that are currently underway will provide invaluable information about both the effectiveness of newly improved anti-A beta vaccines in clinical **treatment**, as well as the role of the A beta peptide in the pathogenesis of the disease.

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